PERIPHERAL PARALYSIS

BY

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ON SOME OF THE VARIETIES OF

PERIPHERAL PARALYSIS.

BY

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PREFACE.

My object in publishing the following pages is to place before the profession the results of my observations upon a large number of cases of paralysis from inflammation of the peripheral nerve trunks that have come under my notice during the past four years. Most of these observations and cases are to be found in a series of Papers read before the Northumberland and Durham Medical Society during the Session, 1886-87, which are now revised and expanded. With the object of curtailing space, I have been careful to select from my note-books only those cases that appeared necessary to illustrate the subject. I have not touched upon diptheritic paralysis, as the subject is already a familiar one.

D. D.

Newcastle-upon-Tyne,
August, 1888.

INTRODUCTION.

The subject of paralysis from neuritis is probably the most important, and certainly is the most recent development of neurological science; indeed, I am inclined to think that here practical medicine has made her most important advance during the past five years. Like myxœdema, Charcot's disease of the joints, and other obscure conditions recently cleared up, it has come upon the profession almost in the light of a new disease. The text-books fail to furnish us with any reliable information; in truth, they are almost silent; and for information on the subject one is compelled to refer to the current medical journals, in which isolated papers are to be found, but more especially to Buzzard's Harveian lectures, published in the *Lancet*, November 28th, 1885. To this author we are indebted for most of the published information; or, to speak more correctly, Dr. Buzzard has collated in the lectures referred to the most important clinical points that go to make up the group of peripheral paralyses. Gowers' latest work, "Diseases of the Nervous System," vol. ii., also contains a good account of periperal lesions. That the cases are numerous there can be no doubt, and the marvel is that they have so long escaped recognition, considering the astute and watchful supervision begotten of the keen competition of the present day. Until recently, the observations upon periphĕral paralysis and neuritis were well-nigh exclusively confined to the various forms of neuralgia, to lesions the outcome of traumatic inflammations, the invasion of neighbouring disease, or accidents, and, of course, to a few well-known forms, such as Bell's paralysis. This category would necessarily include paralysis from pressure exerted by tumours, crushed and divided nerves, and so forth, while the more obscure and the true medical cases were mistaken for examples of progressive muscular atrophy, acute or chronic anterior polio-myelitis, acute disseminated myelitis, locomotor ataxia, hysterical paralysis; and not a few were relegated to, or claimed by, the surgeon as cases of chronic inflammation of tendons or their sheaths, with this disastrous result that limbs, I fear, have been amputated, under the impression that contracted fingers were permanently disabled. And it is only too obvious, from a careful perusal of the weekly medical journals, that this diagnostic confusion is still the order of the day, as cases are frequently reported in which recovery is claimed for serious organic central lesions generally taught to be beyond the aid of our science. Nor is the cause of the confusion far to seek, as a glance at the great variety in distribution as well as degree of the paralysis may easily suggest. Few would be likely to recognize a connection between a very mild and transient attack of Bell's paralysis of the *portio dura* from cold, and a rapidly developed paraplegia, with complete and permanent paralysis and wasting, during the progress of which the patient may succumb. Nor does it appear easy to place in the same pathological category a case of paralysis of the extensors of one or two fingers, and a lesion that paralyses the sensory and motor fibres of nearly all the nerves of the arms and legs, together with producing double facial paralysis and ptosis of one or both lids, and paralysis of the palate. But all these may be examples of peripheral neuritis. Again, there is a marked tendency to depart from the supposed type of a peripheral paralysis, which is often forgotten. Thus, in a large proportion of the cases, the paralysis is confined to the motor nerves, and there is little or no wasting, and the electrical reaction may or may not be altered; for, it must be borne in mind that, when we speak of a typical case (if, indeed, it be wise to seek for type in disease), *pain on pressure: hyperalgesia, with areas of analgesia; motor paralysis, with wasting of the muscles and altered electrical excitability: and more or less vasomotor and trophic disturbances in the skin* (swelling, eruptions, etc.), are generally included. Further, a brief consideration of the causes at work in producing neuritis may serve to emphasize the great difficulty there is in bringing into one group all the numerous varieties to be met with. Unfortunately, our present knowledge will only permit us to grope somewhat blindly in the domain of etiology, but a few of the causes are sufficiently obvious to allow us to speak with tolerable certainty. In my experience, the most common cause of peripheral neuritis is alcohol; then cold and rheumatism, lead, septic conditions, syphilis, gout, diphtheria, and traumatism; but, in a considerable number of instances, it is impossible to give the cause a name. It will at once be recognized how little resemblance there is between an alcoholic and a lead case; the former characterized by paralysis of the legs, with pain in the muscles; whilst the latter is shown by drop-wrist, and a total absence of sensory disturbances. Nor is it an easier matter to group an alcoholic case in which the upper extremities are chiefly affected with a plumbic case, inasmuch as the *supinator longus* may be attacked in the former but escapes in the latter. Thus it appears that cases of peripheral paralysis differ widely, and would almost defy an attempt at classification.

CHAPTER I.

ALCOHOLIC PARALYSIS.

The alcoholic variety of peripheral paralysis deserves our closest attention, and I feel that I am justified in considering it before the other forms, first, because it is the commonest; second, because it is the most dangerous to life and limb; and third, on account of the opportunities that have occurred to me of working at the pathology of the affection.

Few varieties of paralysis are more common, and, when the case is well marked, present more characteristic features than the alcoholic; yet, as there is a want of uniformity, and all cases are not alike, great care is necessary to avoid mistakes in diagnosis. On account of the cerebral symptoms so commonly met with, the case is liable to be looked upon as one of softening of the brain; others again are confounded with acute ascending myelitis; while not a few are mistaken for locomotor ataxia. But perhaps the resemblance between alcoholic paralysis and the group of sub-acute lesions affecting the spinal grey matter, of which the so-called *sub-acute anterior general spinal paralysis* of Duchenne may be taken as a type, furnishes us with diagnostic problems of still greater difficulty.

My observations include twenty cases, of which fifteen were women, and five adult males.* The ages of these patients vary from 24 to 63 years, whilst the great majority are from 30 to 50: though, doubtless, we may expect to meet alcoholic paralysis at any period of life, provided the excessive use of the stimulant has been indulged in. It is a striking and interesting fact that women should be so much more frequently attacked than men, the reason for which has yet to be worked out. It is suggestive, however, that four out of the five men in my list of cases led sedentary lives, and were "soakers" rather than erratic drinkers, as working men so often are. One of them was a tram conductor, one a man of independent means, and two kept public houses. In the manner of drinking, therefore, as well as mode of life, they resemble somewhat the ordinary female alcoholic, who is essentially a chronic toper. Five of the total number of the cases recovered; six were lost sight of before complete recovery could be registered; and nine died. Of the fatal cases, seven were women and two men, but these points will again be referred to when the question of prognosis is under consideration.

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* Since the above was written, several more cases have come under my notice, but, with one exception, they were in females.

As a rule, the symptoms come on gradually, and overtake the unfortunate alcoholic as he or she is imbibing the usual daily supply of the stimulant; though, occasionally, the affection makes such rapid strides after or during a drinking *bout*, as to suggest that the attack began acutely. No doubt in some instances the paralysis develops somewhat suddenly, when the term acute alcoholic paralysis might be justifiable.* The earliest indication of the approach of the paralysis, which generally overtakes the patient when more or less " out of health " from intemperate habits, is usually the feeling of numbness and tenderness in the lower extremities, the feet and calves being first affected in this way. Not unfrequently one leg is attacked some little time before the other. By degrees difficulty is experienced in walking, the effort becomes painful, and the limbs feel heavy, whilst the movements are somewhat shuffling and uncertain. Pains of a more or less characteristic nature are now complained of. These constitute a most important symptom, and, when met with in a case of true paraplegia, may alone furnish almost sufficient data upon which to found a diagnosis. It is perhaps within the mark to say that the patient experiences three distinct kinds of suffering, though all three varieties of pain may not be present at the same time. In the first place, the pains often shoot down the legs into the feet or other part of the lower extremities, when they simulate very closely the lancinating pains of locomotor ataxia; or constant rheumatoid pains are complained of, which are increased by every movement; and, thirdly, there is commonly a soreness and hyperæsthesia in the feet and affected muscles, elicited by pressure, the slightest touch being, in some cases, sufficient to cause the patient to scream out. This latter phase of the sensory disturbance is very suggestive, and it is noteworthy that it is borne with little or no fortitude. In some cases, I repeat, one or more of these varieties of pain may be absent, but the tenderness of the feet and muscles, especially the latter, is nearly always present. It happens not unfrequently that the symptoms develop no further than this degree of paralysis, when, provided the alcohol be withdrawn, the patient may gradually recover. As a rule, however, the motor weakness increases until the patient can no longer walk, or even stand. Along with this increasing paralysis is observed a corresponding diminution in the bulk of the affected muscles, which, in some cases, undergo very rapid atrophy. Those usually first paralysed are the tibialis anticus, the extensor longus digitorum, and the extensor proprius pollicis, or muscles supplied by the anterior tibial nerve. So that the condition which I have ventured to call *ankle-drop* is produced, the foot being extended at the ankle. This condition is an important diagnostic aid, and is certainly highly suggestive though by no means pathognomonic of the form of paralysis under consideration.

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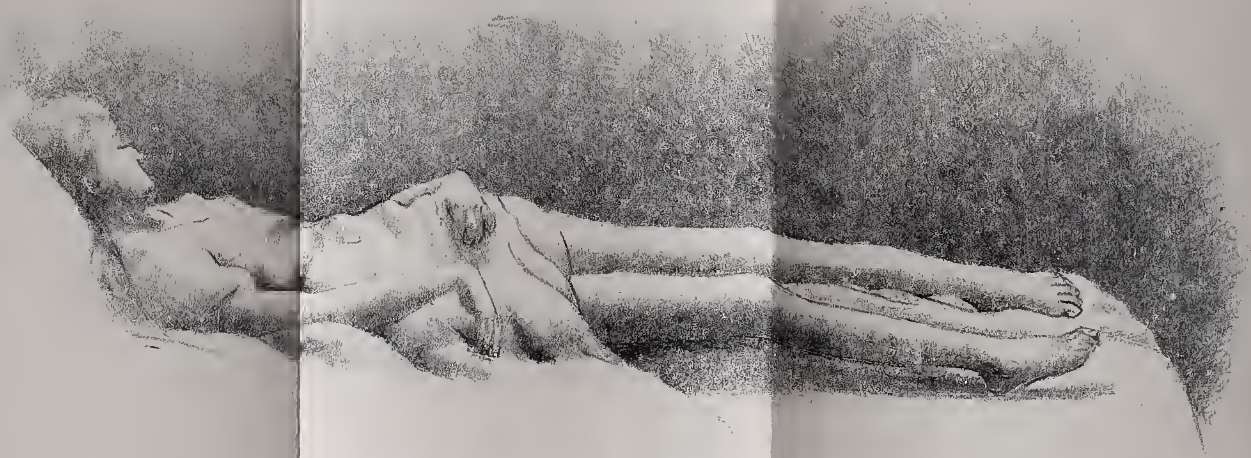
* See reference to a case on page 49.

The muscles on the front of the thigh (quadriceps femoris) are also very frequently paralysed, nor are the calf muscles likely to escape in well-marked cases. The muscles now feel soft and flabby, and are, as I have stated above, exquisitely tender. Every attempt to move, or otherwise interfere with the limbs, is strongly resented. Indeed, in many instances, the patient is as jealous of interference as if he were the subject of acute rheumatism.

Accompanying the atrophy, a marked diminution in faradic contractility is commonly noticed. This is very pronounced in some cases, so much so that the strongest interrupted current may fail to contract the wasted muscles. When carefully tested with both forms of current, especially at a comparatively early period of the paralysis, the *reaction of degeneration* may easily be demonstrated. As this is a point of importance in all forms of peripheral paralysis, it may not be out of place to state, briefly, the nature of the reaction. The electrical change first observed is loss of faradic excitability. In a few days the galvanic reaction also declines, but by degrees (ten days or so from the time that the decline was first detected) the galvanic irritability begins to increase, so that a weak current hitherto insufficient to cause contraction will affect the muscles distinctly. It is further noticed that these galvanic reactions are sluggish in the extreme, and that the most energetic contraction is produced when the current is closed with the positive pole on the muscle. In addition to the pains already mentioned, there are other sensory defects of considerable significance. Hyperæsthesia, no doubt closely allied to one form of pain, deserves first mention. With this we have hyperalgesia, if indeed it be practicable to draw a distinction here in cases of alcoholic neuritis The sensitiveness is usually distributed in areas, and, in slight cases, may be absent. On the other hand, it is important to observe that, in some comparatively rare cases, well-marked alcoholic motor paralysis occurs with little or no sensory disturbance; in other words, some cases are purely motor. Next in importance to the hyperalgesia is cutaneous analgesia (loss of pain), which also is met with in patches, according to the distribution of the multiple lesion; though careful examination will generally reveal impaired sensibility in the region of the distribution of the cutaneous branches of the peroneal nerve, viz., outer surface of the leg, as well as the skin between the great and second toes, *i.e.*, internal terminal branch of the anterior tibial nerve. Again, there may be loss of touch impressions (anæsthesia), retarded sensibility (pain), and perverted thermal sense; and, occasionally, there is a well-marked girdle sensation round the trunk, or even round the limbs. The vaso-motor and trophic disturbances most frequently observed are coldness and cyanotic mottling of the surface of the affected limbs, with œdematous swelling; at times purple ecchymotic stains make their appearance, and, occasionally, in severe cases, bed-sores develop. It is to be borne



ALCOHOLIC PARALYSIS. WOMAN, AGED 30.

in mind, however, that, in the majority of cases, there are no bedsores. The same may be said of vesical and rectal sphincter paralysis; for, as a rule, the patient retains control over bladder and rectum, though, in very pronounced cases, complete relaxation may be met with. At first, before the paralysis is advanced, there may be found a lively plantar reflex, though it is quite usual to meet with an early disappearance of this phenomenon. In some cases the knee-jerk is lost from the first, but I have several times met with it even to an exaggerated degree. Sooner or later, as the case advances, the skin reflexes and tendon-jerk disappear, and it may be taken as a general statement that, in well-marked cases, there is a complete absence of all the reflexes of the lower extremities, and further, the tendon phenomenon will, generally speaking, remain absent long after motor power has returned. On the other hand, as might be anticipated from the nature of the lesion (multiple neuritis), in a few rare examples neither plantar reflex nor knee-jerk are entirely absent throughout the whole case.

On the features of the case becoming more pronounced, we are generally able to discover that the lesion has extended beyond the legs, and has begun to affect the upper extremities as well. (See accompanying drawing, which represents a young woman, aged 30, at present in the Royal Infirmary, Newcastle) According to Buzzard,* the latter are more or less implicated in almost all cases of alcoholic paralysis. He remarks: "Where careful observation shows that the lower extremities are alone involved, the upper extremities being quite normal as regards strength, sensibility, and electrical reaction, it will usually, I think, be found that the influence of alcohol may be put out of the question." Whilst admitting that the hands commonly are involved, I am convinced that they sometimes escape; indeed, I have no hesitation in asserting that I have met with some undoubted cases of alcoholic paralysis in which the lesion was confined to the lower extremities. When the arms are affected, the same motor and sensory disturbances as I have described as occurring in the legs make their appearance, only in a slighter degree. Thus we have paresis in the arms and hands, commencing with numbness and advancing to paralysis and atrophy. The muscles first and most profoundly affected are usually the extensors of the fingers. In the slightest cases, some numbness, with incomplete paralysis of the extensors of one or two fingers, may be all that indicates an extension of the affection to the arms. In other cases there is complete drop-wrist, when, as a rule, the supinator longus will be found to be paralyzed. Most frequently the skin is more or less analgesic, and the muscles are sensitive on pressure; but the sensory disturbances seldom approach in severity the analogous state of the lower extremities. In some cases, the arms suffer more than the legs; indeed, the latter may show little or no defect; but as this is an exceedingly rare feature, I shall, further on, quote a well-marked example.

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*Paralysis from Peripheral Neuritis, page 94.

Some cases of alcoholic neuritis display a striking resemblance to tabes dorsalis, for not only is the knee-jerk absent, but there may be shooting pains and marked ataxy. To the question of differential diagnosis I shall return later, but I desire, before leaving this part of the subject, to emphasize the fact that inco-ordination may be the most prominent motor phenomenon during the earlier part of the case. Thus, the patient may fail to recognize the position of his legs in bed; he may be unable to place the leg, with the eyes shut, in any required position—the heel of one foot on the toes of the other, for example; and he may be unable to walk or stand with the eyes closed, and so forth. Though the ataxy is often sufficiently striking in alcoholic cases, I must say that the cases I have met with of neuritis in which inco-ordination has been most marked have not been of this variety.

It is common to meet with certain contractures in peripheral neuritis, and alcoholic cases are by no means exempt from this motor impairment. Thus, during the later stages of the disease the legs may be flexed at the knees by rigidity of the hamstrings; or, again, the feet may be forcibly extended through contraction of the tendo achilles; or, as occasionally occurs, the condition of the hands, known as the *main en griffe* of Duchenne, may be well marked. This latter deformity I have much more frequently seen in non-alcoholic forms of peripheral paralysis than in the form under consideration, though contractures of the lower extremities are by no means rare in alcoholic paralysis.

Dr. Hadden has observed nystagmus in some cases, but I believe this to be an extremely rare feature of chronic alcoholism.* All observers have been struck with the mental condition that so frequently accompanies alcoholic paralysis. Of the twenty cases referred to, in only three was I able to satisfy myself that no mental deficiency existed. In some it was well-marked, while, in others, it had to be sought for with care. There is generally loss of memory, which, in the female, almost invariably extends to her habits of intemperance. Irritability, with general mental hebetude, is seldom absent, and, in some cases, cunning and distrust of near relations are engendered, whilst there may be delusions.

Before proceeding to discuss the pathology and diagnosis, I may, with advantage, adduce one or two examples of alcoholic paralysis which, I think, will be found to bear out the above short clinical description of the disease.

Case I.—Emma D., aged 36, married, the mother of two children, of no special occupation, was admitted into the Infirmary, on September 24th, 1884, complaining of loss of memory and paralysis of the lower extremities. She stated that until her marriage she had enjoyed good health. After the birth of her first child, she suffered from peritonitis, and a similar attack followed the birth of the second. She had two miscarriages, but, in both instances, the fœtus was living. Two years before admission to Hospital she had a slight attack of paraplegia, with pains in the legs, symptoms which lasted about twelve months, when she recovered completely. She has been subject to bilious attacks, with headache, for some years. She admitted that she had acquired intemperate habits, and owned a partiality for beer, though she occasionally drank spirits. For many years she had led a very sedentary life. The illness for which she was brought to the Infirmary began two months before she came under my notice. At first she complained of numbness in the soles of the feet, with a feeling of "pins and needles," and then pain was felt in the calves of the legs when she walked. By degrees, the legs became weak, and walking grew more and more difficult; the knees frequently gave way under her, and the weight of the body gave rise to pain in the feet. Later, a sensation of constriction developed round the legs below the knees, and the tingling of the soles of the feet gave place to the feeling as if they were padded with hair. Soon the weakness of the legs became so pronounced that she was obliged to take to her bed. The memory, which was from the first very defective, failed her almost entirely, especially for recent events, and giddiness and sleeplessness added considerably to her troubles. When examined after admission, the following facts were noted :—Of medium height and build; is pale, and slightly cyanotic about the lips; memory very bad, is easily led to contradict her previous statements. She is unable to walk, and generally lies on her side, with the legs drawn up and flexed at the knees; T. 99°; pulse 80, regular, very weak. Complains of tingling in the soles of the feet, and of a girdle sensation round the calves. Pressure on the soles produces a painful burning sensation which appears to be superficial. Squeezing the muscles below the knee – anterior tibial and calf regions—produces great pain. Attempts at straightening the legs, both active and passive, are very painful, and this is especially referred to the quadriceps muscles She is much troubled with giddiness, headache, sickness, and sleeplessness. The appetite is bad, the tongue is coated, and the bowels are constipated. Tactile impressions diminished in areas, and painful impressions lost here and there on the surface of the feet and legs. There is marked motor paralysis with wasting; the muscles most affected are the quadriceps femoris and those of the anterior tibial region. The feet are extended at the ankle. Plantar reflex and knee-jerk entirely absent. Abdominal reflex marked on the right side, absent on the left. No rectal or bladder disturbance; no bed sores; reaction of degeneration imperfectly marked, viz., complete loss of reaction with the interrupted current, and a very feeble contraction on the application of a slowly interrupted but strong continuous current. Slight paralysis of the extensors of the upper extremities could be made out, but there was no loss of electrical excitability, and but slight tenderness of the muscles on pressure, and no sensory impairment. This patient made slow but steady progress towards recovery. The first distinct signs of improvement were in the direction of loss of pain, the muscular pain lasting longer than the superficial cutaneous hyperæsthesia of the soles of the feet, whilst the pain felt on attempting to extend the legs disappeared before that caused by pressing the paralysed muscles. Soon after the hyperæsthesia vanished the ordinary sensibility of the skin returned, and, on the 28th of November, it was noted that the tactile sensibility of the skin of the legs was normal, and that painful impressions were only slightly diminished. Shortly after this, motor power had so far returned that the patient could walk a little by herself. Then the plantar reflex appeared, and, for a time, seemed to be somewhat increased; but the knee-jerk remained absent all the time the patient was under observation. She was discharged well-nigh recovered about four months after her admission. This case furnishes a good example of a moderately severe attack of alcoholic neuritis that ran a favourable course.

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* See case on page 34.

I shall now give a brief account of a still more pronounced case in which death resulted from inflammation of the lungs. The patient was placed under my care by Dr. Slater, of Newcastle, who kindly supplied me with the earlier details of the case. A widow, aged 58, the mother of eight children, complained from time to time, for many months, of dyspepsia, accompanied with vomiting, and rheumatic (?) pains of the feet and legs. By degrees her legs became numb and powerless, and walking was interfered with. When she first came under observation in August, 1886, she was confined to bed; her feet were swollen and pitted on pressure, and were extended at the ankle, though slight flexor movements remained. The arms were also affected, and the muscles of both upper and lower extremities were soft and flabby, and extremely tender to the touch, indeed, the slightest pressure on the anterior tibial and calf muscles caused her to scream. The feet were likewise very tender, and passive movements caused severe pain about the knees and ankles. Knee-jerk and plantar reflex were absent. There was anæsthesia—especially loss of painful impressions—on the dorsum of the feet. Soon the ankle-drop became complete, whilst the power of flexing and extending the legs was lost. The paralysis of the extensors of the hands and fingers then increased, and drop-wrist developed. She now lost control of the bladder and rectal sphincters, and suffered greatly from thirst and constipation. The memory was very defective, and occasionally she showed a tendency to delirium. The paralysed muscles atrophied rapidly, especially those of the anterior tibial region—tibialis anticus, for example, and electrical (faradic) excitability disappeared. The pupils were contracted, and responded to efforts of accommodation but not to light. She steadily grew more and more feeble, and a bed sore developed. On the 5th of October pneumonia set in, and the patient died on the 8th. Permission could only be obtained for a very limited *post-mortem* examination; however, portions of the great sciatic, anterior tibial, and musculo-spinal nerves were obtained for microscopical examination. It should be stated that this patient had been, for some years, addicted to habits of intemperance; whiskey was her common stimulant.

The following case is of great interest, as it tends to show that in alcoholic paralysis the arms may, in some rare cases, bear the brunt of the poison :—

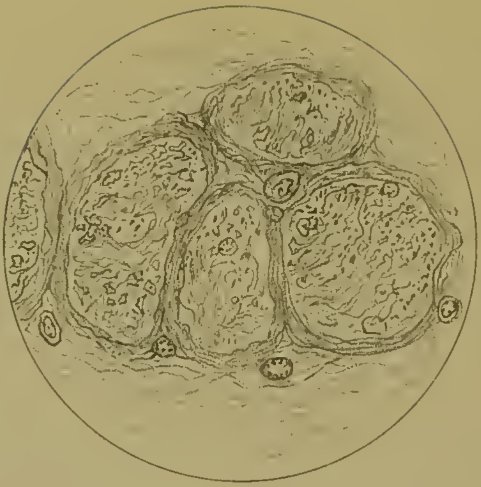
George F., aged 43, a publican, was admitted into the Infirmary, in October, 1886, complaining of loss of power of both arms. He was a heavy drinker. His illness commenced eleven months before with pains in both feet, which, for a short time, interfered with walking. A month subsequent to the commencement of the attack he had an epileptic fit. Soon the pains left the lower extremities, and lodged in the shoulders and arms, and, eight months before his admission, the arms became weak and paralysed, the weakness beginning in both arms at once. At first, the pains and paralysis affected the deltoid regions particularly, so that he could not raise the arms. About a fortnight before I first saw him drop-wrist developed, and the muscles on the back of the forearm rapidly wasted. When examined, it was noted that he could walk without difficulty. The legs were fairly developed, and, indeed, appeared to be normal, except that the knee-jerk and plantar reflex were absent. Both arms were markedly paralysed and atrophied, especially the deltoids and extensors of the wrists and fingers. There was complete double drop-wrist, though the wasting and paralysis were rather more pronounced on the right than the left side. The muscles were tender. Analgesia and anæsthesia were present in circumscribed areas. The deltoids and the muscles supplied by the musculo spiral were most affected, including the long supinators. The reaction of degeneration was marked in the musculo-spiral region. This patient, unfortunately, left the Infirmary the day after his admission, and I have not been able to trace the case.

I now come to an undoubted alcoholic case in which the right upper extremity was the only paralysed limb. C. C., aged 44, a widow, came under observation two weeks after the symptoms of paralysis appeared. It transpired that her husband died a short while before her illness, and it was concluded, with an efficient show of reason, that in order to drown her grief she had indulged her intemperate proclivity (for there was a strong alcoholic history) more than usual. The attack began with a feeling of numbness in the right hand, but especially about the ends of the radius and ulna. Then, in rapid succession, paralysis of the extensors and flexors of the fingers occurred. On examination, the hand was observed to hang as in lead palsy, and the power of grasping was exceedingly feeble, in fact, was entirely absent on the ulnar side. The supinator longus was paralysed, as were also the muscles supplied by the ulnar and median nerves, whilst, in addition, the triceps and biceps were implicated, though to a less extent. The affected nerves were the musculo-spiral, ulnar, median, and musculo-cutaneous. With the exception of the triceps and biceps, all the paralysed muscles were considerably wasted, and showed electrical changes suggestive of degeneration.

There was little or no pain complained of; indeed, there was comparatively slight sensory disturbance of any kind The left arm and both lower extremities, reflexes included, were normal. This patient made a tedious recovery, but some months later, succumbed to a large dose of infusion of match-heads, taken when intoxicated, with intent to commit suicide, when early cirrhosis of the liver was discovered, most probably alcoholic. When considering the pathology of the case, I had some considerable difficulty in disposing of injury from lying on the arm, which naturally suggested itself to one's mind, but as the result of careful enquiry, and from the distribution of the paralysis, I concluded that I was justified in putting this view out of court. Of course, the case must be regarded as quite exceptional.

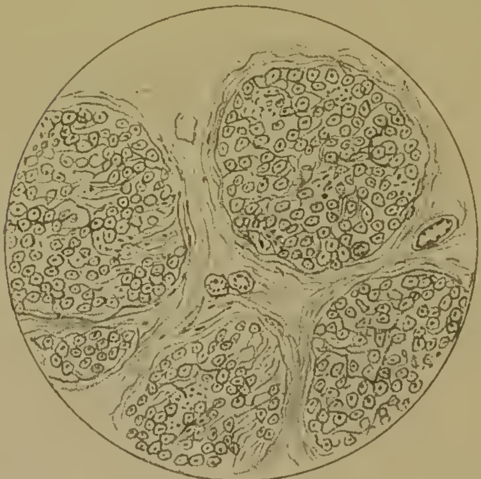
I now turn to the subject of the pathology of alcoholic paralysis, and although I have, without apology or argument, ventured to include the disease in the category of cases of peripheral neuritis, it is important to recollect that, at times, the spinal cord has been found diseased when alcohol alone was responsible for the paralysis. This fact, however, does not invalidate the statement that the symptoms I have described are now generally believed to depend upon a lesion of the peripheral nerves I have had the opportunity of examining the nerve trunks of two cases, and I shall briefly describe the changes noted. To Dr. Gowans I am indebted for this opportunity in one instance, as he very kindly furnished me with the details of a case, and allowed me to take part in the *post-mortem* examination. The patient was a woman, aged 42, who died with typical alcoholic paralysis complicating cirrhosis of the liver; both arms and legs were paralysed, although the latter, in accordance with the usual experience, were decidedly the most affected. We examined the following nerves:—Great sciatic, internal popliteal, external popliteal, musculo-spiral, and ulnar, and in all more or less morbid conditions were found. The most striking changes were observed in the sciatic, and these, generally speaking, were characterised by alteration of the nerve fibres (parenchymatous neuritis), though, in places, there was distinct increase in the interstitial substance, especially between the

FIG. I



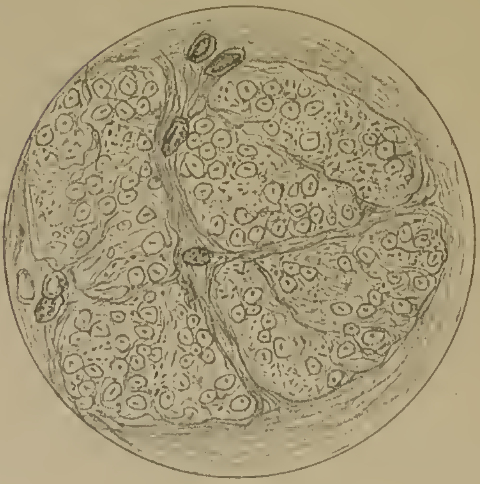
ALCOHOLIC PARALYSIS—ANTERIOR TIBIAL NERVE.

FIG. II.



MEDIAN NERVE (NORMAL).

FIG. III.



LEAD PALSY—PORTION OF THE POSTERIOR INTEROSSEOUS NERVE.

fasciculi. In the smaller nerves, the lesion appeared to be confined almost entirely to the nerve fibres, and seemed to be chiefly degenerative. Where the parenchymatous inflammation was intense the axis-cylinders were markedly changed. In places they were swollen and hypertrophied, or else they had disappeared or were atrophied. The white substance of Schwann was here and there conspicuously granular and broken up, so that masses of myelin appeared to be isolated from the fibres. In conducting the microscopical examination, the difficulty of distinguishing between the processes of inflammation and degeneration was necessarily felt, though pronounced interstitial changes, dilatation of blood vessels, and multiplication of nuclei were fairly assumed to indicate an inflammatory lesion. The sciatic, anterior tibial, and musculo-spiral, along with the paralysed extensor longus digitorum muscle of the fatal case, the particulars of which I have related, were also examined. The anterior tibial showed much greater disorganization than the other nerves, indeed, there was scarcely any healthy nerve tissue seen in the sections examined. The morbid changes were largely parenchymatous, though the nuclei of the endoneurium were proliferated, and the walls of the vessels considerably thickened. The muscle showed little that could be found fault with. Here and there, perhaps, the fibres were slightly granular, and the cross striation less pronounced than in health, but lying quite adjacent were fibres in which not the slightest departure from the normal could be recognised, and this was the rule.

It is undoubtedly difficult to explain the simultaneous affection of so many peripheral nerves, the centres remaining free, in alcoholism and other conditions wont to give rise to neuritis; and, as is well known, it has been suggested that, although it has often escaped recognition, there must after all be some central disturbance responsible for the widely distributed lesion. Notably Erb and De Watteville have advanced the view that a functional disturbance of the motor ganglion-cells of the cord may occasion degeneration of the motor fibres continuous with the anterior roots. Buzzard, on the other hand, has suggested the hypothesis that the changes in the peripheral nerves may be caused by altered blood supply, through interference with the vaso-motor centres in the medulla and cord. Without venturing to offer an opinion upon these suggestions, I think, in considering the way in which the multiple lesion is produced, due stress must be laid upon the fact that the *materies morbi* is circulating in the blood—the poison of rheumatism, alcohol, the morbid products arising from chill, for example. And again, it must not be forgotten that the appearances are often those of active inflammation rather than degeneration. But, perhaps, the most damaging argument that can be advanced against the suggestion that the nerve trunk lesions are secondary to toxic disturbance of the trophic cells of the cord is to be found in the fact that in the great majority of the cases the anterior roots escape. It would appear to be a fair explanation of the ataxy so commonly met with in alcoholic paralysis to assume that in some cases the muscle-sensory nerves are chiefly implicated, for it is unnecessary for me to remind the reader that to such a disturbance is largely due the inco-ordination of tabes, though, in the latter case, the block occurs in the root zone of the cord.

The diagnosis of alcoholic paralysis is seldom difficult, unless the more typical symptoms are absent, and intemperance is absolutely denied. The early pains may be mistaken for rheumatism, though their situations scarcely correspond; and again, the tenderness of the feet, and feeling of needles and pins in the legs generally, would suggest alcohol. The diseases of the spinal cord with which this form of paralysis might be confounded are numerous, but the greatest diagnostic difficulties arise in connection with acute and sub-acute lesions of the cells of the anterior spinal horns and locomotor ataxia. The onset of the attack, which, as we have seen, is usually ushered in by pains, will greatly aid in eliminating polio-myelitis; nor would we expect to meet with anæsthesia in that disease; and, further, the distribution of the paralysis is nearly always symmetrical in multiple neuritis, whereas, in the spinal lesion, it is often erratic. It is also important to recollect that the order in which the muscles are paralysed is different in the two diseases. In alcoholic neuritis, the first muscles of the lower extremities to be affected are those supplied by the anterior tibial nerve, especially the flexors of the foot at the ankle, then the extensor of the knee ; and, in the upper extremity, it commences by attacking the extensors of the wrist and fingers. On the other hand, in the spinal affection, the following order is usually observed :—feet, leg, flexors of thighs, and extensors of thighs, and, in the arm, the small muscles of the hand are affected before the extensors of the forearm. The most important point to be attended to in distinguishing between tabes and alcoholic paralysis are muscular hyperæsthesia, paralysis of the ankle flexors, and atrophy, with loss of electrical excitability, all common features of the latter disease. Nor are mental symptoms often observed in locomotor ataxia.

CHAPTER II.

Progressive Multiple Neuritis.

We have seen that the effect of alcohol upon the nerve trunks is fairly uniform, the legs being first and most affected, and the arms later and to a less extent. In the group of cases that will next occupy our attention the neuritis is frequently of wider distribution, and may attack the cranial nerves in the course of its development. It is essentially a poly-neuritis, and, pending the discovery of its true pathology, for at present we know little or nothing of the etiology of the disease, it may be named progressive multiple neuritis, though, to some cases, the term *universal* might be aptly applied.

The following case, which I saw with Drs. Hume and Blacklock, is a good example of the universal form of neuritis:—O. G., age 25, first complained of feeling ill on the 13th February, 1884. He had been in a draught in church a day or two before, and ascribed his illness to catching cold. His attack began with loss of appetite and a feeling of constriction across the chest. In a short time this feeling amounted to pain and descended to the stomach. Then vomiting set in, and the pain went round to the back, where it remained for the next few days. With the backache the sensation of tingling, like needles and pins, in the legs and arms was felt, especially in the former, which rapidly became powerless. By the 21st, eight days after the commencement of the symptoms, he was so far paralysed that he was obliged to remain in bed. After this the paralysis rapidly spread upwards and affected the arms, especially the left, so that by the 25th of the month both arms and legs were nearly helpless. My connection with the case dates from this period. The legs were flaccid and almost completely paralysed, and the feet were extended at the ankles. There was little or no diminution in the bulk of the muscles, which were tender on pressure. Sensibility was impaired in patches, but here and there areas of hyperæsthesia were detected. The plantar reflex and knee-jerk were absent. The arms were similarly affected, though to a less extent, as considerable grasping power still remained, especially on the right side, nor was control entirely lost over the flexors of the forearms. Thus, in both upper and lower extremities, the extensors were first affected. The back-ache continued more or less, and the feeling of constriction round the chest, which had subsided, once more was complained of, giving rise to a difficulty in breathing. He now called our attention to a strange subjective sensation which he felt all over the body, but especially in the chest, abdomen, and legs—the feeling that the skin had become hard, leathery, and insensitive. The tongue was coated, and the appetite bad. The temperature and pulse were practically normal. About three weeks after the commencement of the illness, marked constipation set in, and enemata had to be used, and he lost power over the bladder, which had to be evacuated by means of the catheter. The paralysis of the arms and legs increased, and before long it was apparent that the muscles were undergoing rapid atrophy. On the 6th of March, he began to complain of pain in the feet and legs, especially the right. This increased, and in a day or two became most intense, and indeed continued so, more or less, up to the end of the sixth week of the illness. The pain was almost constant, and was of a sore, aching character, the feet being exquisitely sensitive to touch. The only remedy that afforded him any relief was morphia, administered hypodermically. Towards the end of March, about the seventh week of the illness, the facial muscles became affected, and, in a short while, double facial paralysis developed, the muscles supplied by the upper divisions of the portio dura being implicated in the process. It was also observed that his speech was thick, and that swallowing was interfered with. An examination of the mouth showed paralysis of the left side of the palate, which hung flaccid during a deep inspiration. The arms and legs were now markedly paralysed and wasted, the facial muscles were motionless, respiration was interfered with, and the catheter had still to be employed. This distressing condition of things did not last long in its entirety, for shortly after the appearance of the facial paralysis an improvement became apparent in the arms and, a little later, in the legs. Notwithstanding this partial return of motion in the legs they gradually became drawn up and once more the paralysis appeared to increase; they then remained *in statu quo*—drawn up and paralysed—until the end of June, when they gradually lost the stiffness, and power began to return. During the last few weeks of this period, a marked improvement took place in our patient's general health. He could eat well, could sit up, the facial paralysis disappeared, and, by degrees, recovered the use of his hands. The lower extremities, however, recovered very slowly, and it was long after he was able to drive about in a spinal carriage that any considerable improvement was noticed in them.

Early in the case, the paralysed muscles were tested electrically, when the reaction of degeneration was apparent.

About August, 1885, 18 months from the commencement of the attack, he abandoned crutches for two sticks, and when last I heard of him (January 27th, 1886) he was able to walk about without any support, almost as well as ever, but when he attempted to run, he felt that he was in danger of falling. He wrote me at that time, for he left Newcastle for the country as soon as he was able to be moved, stating that "the toe-joints were still a little stiff, and the sinews of the ankles appeared to be drawn rather tight, and the feet were more sensitive than natural." I may add that no history of syphilis could be obtained, and that the patient was practically a total abstainer, so that alcohol could not be held responsible for the paralysis.

The following case is interesting as showing that multiple neuritis may, at least for a time, have an hemiplegic distribution. The patient, whom I saw with Dr. Watson, of Newcastle, was 35 years of age. His illness dated from June, 1882, when he began to complain of the feeling of weakness, which affected the whole body more or less, but especially the left side—

arm, leg, and face. The fingers of the left hand then became somewhat stiff and flexed: but notwithstanding the hemiplegia, he could walk about. During this attack, which lasted three months, he was pale, and felt very weak. In September, 1883, a few weeks before his marriage, the paralytic symptoms threatened to return. This time they began with diminished control over the sphincter of the rectum, and then the left foot became weak and unmanageable, "it flopped down in a clumsy way when he walked." Then the left side of the face became paralysed, and soon the muscles of the right side of the face began to twitch. From this the weakness extended and affected both arms and legs, though the onset was very gradual, for it was not until the beginning of March, 1884, that the paralysis had developed to such an extent as to confine him to bed. He was now unable to write, as his fingers could not grasp the pen. The auditory nerves became affected, and by the end of April he was "stone deaf." Taste on the left side of the tongue was lost, and double vision (distinct squint), double facial paralysis, difficulty of swallowing, all developed. On the 22nd of April, 1884, when the attack was at its height, the following condition was noted: arms and legs paralysed and wasted, the feet extended at the ankles, deafness, squint, paralysis of the left side of face and partial of the right side, unable to protrude the tongue, speech very thick, loss of taste (left side), unable to empty the bladder—the urine dribbling away when the viscus became full. rectal sphincter relaxed, threatened with a bed-sore. I must add that he experienced no pain, and that "reflex action" was marked in the legs.* By degrees the various paralyses improved; the cranial nerve lesions disappeared soonest, and then the weakness of the arms passed off, leaving a degree of impairment of the legs and sphincters, for which I was asked to see him.

He could walk about, though with obvious difficulty, the left leg being dragged more than the right. The muscles were flabby and somewhat wasted, and showed a decidedly diminished electrical excitability (quantitive loss), especially those of the anterior tibial region. The knee-jerk was increased on both sides, though particularly active on the right, but the plantar reflex was absent. Sensibility was almost restored.

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* I did not see the patient at this time, nor was he under the care of Dr. Watson until later, so that we could not procure precise details as to sensibility, reflex action, etc., but the patient, who was a gentleman of great intelligence, furnished his history with striking clearness.

The points of interest in this case are the tendency of the paralysis, at first, to limit itself to one side (hemiplegic), and secondly, the peculiar behaviour of the so-called deep reflexes, which were increased, instead of diminished or lost. Then it is interesting to note the extent to which the cranial nerves suffered—there was deafness, loss of taste, squint, and facial paralysis. There was no history of syphilis or of alcoholic excess.

In the case I am about to relate, the hemiplegic distribution was also, to a great extent, preserved, and the afferent nerves appeared to suffer more than the motor. The patient, a medical man, consulted me for numbness of the left hand, and I give the history of his case in his own words, which are accurately descriptive of his attack:—"On Thursday, June 23rd, I first noticed a tingling between the outer side of the ring. and the inner side of middle finger of the left hand, as if some pure carbolic acid had been spilt upon them. This continued, but I thought little of it until the Saturday, when the feeling of numbness extended over the whole hand and up to the forearm. On Sunday, my left foot tingled in the same way. The next day, as it had extended to my leg, I consulted Dr. Drummond, and on examination, he found the sense of touch normal, with the exception of a circular patch on the dorsal surface of the hand, near the knuckles, which was anæsthetic; motor power and co-ordination were pretty fair. On Tuesday, I was able to drive, but the tingling now affected the whole side, including the neck, but not the face. I may say here that previous to the above symptoms, I had been subject, for some time, to startings at night just as I was on the point of going to sleep; I now observed that these startings increased. On Saturday, July 2nd, my left wrist became painful if not supported by the other hand. Now the power of co-ordination rapidly left me.

At the beginning of the next week, when I was again examined, the sense of touch was found to be much impaired, for I could not tell that anyone was touching my hand, even when my fingers were tightly squeezed, and if I lost sight of my hand I could not find it again until I saw it. Above the wrist sensation was pretty good. During the week I lost all control over the hand, the extensor muscles being chiefly affected causing the hand to drop and the fingers to assume the claw-shape. At no time was there complete loss of motor power, only the muscles were weak, so that though I could extend the hand at will I could not sustain the effort for more than a very short period. There was also complete loss of co-ordination. My speech was slightly affected about this time as regards words of three or more syllables, that is, I could not be certain of pronouncing them correctly without two or three attempts, and then they were clipped, but that soon passed off. I also had a pain (or more of a constriction) across the abdomen, on a level with the umbilicus after meals and immediately before passing a motion. Friday in the same week (July 8th) I went to bed, where I remained for three weeks. During the first week, the tingling of the neck and side of body disappeared, but the other symptoms remained much the same. The sensations of my hand were very eccentric. Often I imagined it to be in one place when it was really in another. During the second week I lost all the tingling of the leg and foot, but had it sometimes in the right hand and foot. The left hand and arm now improved, and I rapidly acquired control over its movements. By the end of the third week, my wrist was still painful if not supported, but the sense of touch was improved. I could now describe pretty accurately where my fingers were touched, but not so well on the back as on the front of the hand. I had very slight power of grasping, being unable to hold a fork at meals, or dress myself with that hand for sometime after. On first attempting to walk again, I was rather unsteady in my gait, and could not go more than a hundred yards at first without feeling severe fatigue, and a pain at the back of the knee, chiefly over the ext. popliteal nerve, but they passed off with rest. The tingling had left the arm and forearm, and seemed to be worse over the distribution of the ulnar nerve in the hand, the same occurring on the right hand, but not so bad. From that time I have gone on steadily improving, and now I can walk three or four miles without a rest. The tingling still affects the palmar surface of the left hand and fingers, and, after much exertion, or use of the right hand, I can always feel it in its palmar surface as well, and sometimes on the soles of both feet."

At this stage, I again had an opportunity of examining the patient. I found comparatively little evidence of the paralysis of the extensors of the left hand, though the muscles reacted very feebly to the interrupted current, and there was still some wasting.

The inco-ordination of the left arm during the height of the attack was very striking, and evidently pointed to an extensive lesion of the afferent muscle nerve fibres. Here, again, I was at a loss to account for the neuritis, as lead, alcohol, syphilis, gout, and rheumatism, had obviously nothing to do with the condition.

In some cases of multiple neuritis gross inco-ordination and ataxy, more pronounced even than is met with as a rule in cerebellar disease or tabes dorsalis, is the most striking feature. In the last case referred to the ataxy of the left arm was complete, but in the case of the patient, whose history I am about to record, the lower extremities were chiefly affected. This case is interesting from many points of view, but I adduce it more especially with the object of showing that peripheral ataxia is not invariably due to alcoholic poisoning, an impression that is certainly conveyed in a comparatively recent utterance of Professor Charcot.* The patient, whom I saw with Dr. Park, of Houghton-le-Spring, was a young married lady. For upwards of two months before she came under my notice she complained of numbness in both hands, commencing in the left, and latterly affecting the lower extremities as well. This sensation in the feet and legs rapidly developed into a want of confidence in walking, and loss of control over other muscular movements, so much so, that her friends detected her in staggering from time to time. She stated that going up and down stairs was the most difficult action. She frequently fell when walking, and, of late, had observed that she was unable to use her fingers in buttoning her gloves, tying strings, etc.; but she experienced most difficulty in lifting a cup to the lips, as the contents were spilt unless she watched the movements of the arm closely. There was no complaint of pain in the back or limbs, and, with the exception of the motor difficulties, she considered that she was in good health.

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* *British Medical Journal*, January 1, 1887.

I found her robust in appearance, and exceedingly intelligent. When she attempted to walk across the room she staggered and kept her eyes fixed on the floor after the manner of an ataxia patient. It was also apparent that in walking she scraped the toes of the left foot as she brought the leg forward. There was no wasting or definite paralysis of the muscles of the lower extremities, though she had some difficulty in flexing the left foot at the ankle; nor was there any anæsthesia of the legs, or impairment of the electrical excitability. The knee-jerk was absent on both sides, but strange to say, slight ankle-clonus could be elicited, and the plantar reflex was increased. In the upper extremities I found the grasping power diminished, especially of the left hand, and, on both sides, there was loss of power in the extensors. The left hypothenar muscles were somewhat wasted, and an attempt to extend the fingers fully gave the hand the claw-like appearance, so characteristic of ulnar paralysis. In addition, there was some anæsthesia of the hand in the region of the distribution of the ulnar nerve.

A month later, I again had an opportunity of examining the patient. The general health remained good, indeed seven pounds weight had been acquired during the month. She stated that during the first fortnight, an improvement was manifest in the paralytic condition, but subsequently, the difficulty in walking increased. An examination revealed the fact that she was decidedly worse. In attempting to walk, the left foot was scraped along the floor, and it was apparent that she had a great difficulty in flexing the foot, leg, and thigh. There was no wasting, unless it were of the anterior tibial muscles, which were weak and flabby on both sides. *The knee-jerk and ankle-clonus of both legs were very pronounced. The plantar reflex was absent.* Locomotion was shaky and uncertain owing to a great tendency to staggering. She was unable to stand with the feet wide apart when the eyes were closed. On carefully testing the strength of the muscles of the legs, the flexors of ankle, knee, and thigh, were found to be weak on both sides, but the power of extension remained good. A small area of anæsthesia, the only impairment of the sort, was detected on the inside of the calf of the right leg, about four inches below the knee. The extensor paralysis of the hands and wrists was more apparent than before, and the thenar and hypothenar eminences were wasted. Tested with the faradaic current the wasted muscles showed a decided quantitive loss. The anæsthesia was also increased and now affected the region of the median, as well as that of the ulnar of both hands.

When I again had the opportunity of examining this patient, some months later, I found a marked improvement, which I believe has continued, for I understand she is now able to walk about, and, indeed, is practically in good health.

The cases of progressive multiple neuritis just referred to resemble somewhat closely the account given by Hirsch, in the Hand-book of Geographical and Historical Pathology, of the disease met with in Japan and described under the name of kakké or beriberi. There are two forms of this disease, the paralytic, dry, or atrophic variety, and another which is characterized by the presence of dropsy, in addition to the paralysis. The former would appear to show itself by a gradually increasing paralysis (sensory and motor), with muscular wasting and pains, commencing usually in the lower extremities, and attacking, later, the arms, but in acute and severe cases extending over the whole body. Unlike the examples of multiple neuritis I have recorded, in the cases of endemic neuritis of Japan, and other parts of the East Indies, there are marked disorders of the blood-forming organs giving rise to prostration and anæmia, and, in many cases, the nervous symptoms are of decidedly less importance than the general blood state.

The diagnosis of the non-alcoholic form of multiple neuritis should not be difficult now that the existence of such a disease is so generally recognised; except, perhaps, in some special cases, as, for example, when the distribution of the paralysis is hemiplegic, when cerebral disease might suggest itself. The progressive paralysis with wasting, attacking muscle after muscle, and attended by loss of tendon-reaction and skin reflexes, make up a suggestive picture, especially if the electrical excitability is either lost or markedly modified. It is well to recollect, however, that the muscles do not always waste to any striking degree, that the myotatic irritability and superficial reflexes are not always absent, and that the electrical reaction is not invariably characteristic. Indeed, it would appear that a too rigid belief in the immutability of the recognised type of peripheral paralysis has led many an observer into error. I have already referred, when dealing with the diagnosis of alcoholic cases, to the order in which the muscles become paralysed in neuritis, as compared with spinal lesions, and I may be allowed once more to emphasize the fact that, in peripheral paralysis beginning in the lower extremities, the muscles that flex the foot at the ankle (anterior tibial group), are usually first attacked, then the knee extensors. Whereas, in chronic and sub-acute cases of atrophic spinal paralysis, the march is often feet muscles, calf, anterior tibial group, flexors of knee, and so forth. Again, peripheral lesions attacking the upper extremities most frequently show themselves first by paralysis of the extensor group of the wrist and fingers, whilst the spinal disease affects primarily the small muscles of the hands.

I have observed that we must not always look for marked wasting, nor for the formula of the reaction of degeneration, or even for diminished electrical contractility and loss of tendon-jerk, and, I would add, impairment of sensibility, as I have met with undoubted cases in which the more common features of peripheral paralysis were well-nigh reversed, of which the following is an example:—

A. J., aged 24, a baker, was admitted into the Royal Infirmary for paralysis of the right arm and face. Six months previously, when in a semi-intoxicated condition, for he was much addicted to alcohol, he fell and struck his right elbow. The blow was evidently severe as an extensive bruise appeared at the back of the joint. In a week there was complete wrist drop. Then he noticed that he was unable to grasp with the right hand, and that both arms were slightly affected by tremor. At the time of his admission, the right arm and hand were paralysed, the wrist and fingers were dropped as in lead palsy, and the flexors were decidedly weak. The supinator longus was paralysed and atrophied, but, with this exception, there was little or no diminution in the bulk of the paralysed muscles. The small muscles of the hand supplied by the ulnar did not appear to be materially affected. The sensibility of the limb was practically unimpaired. A tremor, often scarcely appreciable, but, at times, fairly marked, was noticed in both hands, especially when a muscular effort was made. The left arm was somewhat weaker than in health, but there was no definite paralysis, and the same may be said of both legs. The knee-jerk was very lively on both sides. The plantar reflex was slightly marked, contraction of the quadriceps being especially noticeable. The face was paralysed on both sides, but the impairment was incomplete, as grimaces could be made in a feeble and tremulous way. There was very pronounced lateral nystagmus. All the affected muscles responded to weak, continuous, and interrupted currents, and this heightened contractility increased as motor power returned.

This patient is at present under treatment and promises to do exceedingly well. I believe, in his case, the alcohol operated as a predisposing cause, whilst the injury determined the attack of neuritis. The diagnosis was necessarily a matter of difficulty, as the tremor and nystagmus forced disseminated sclerosis into the consideration. However, the distribution of the paralysis (one arm and both sides of the face), as well as the arm muscles affected (musculo-spiral group, small muscles of the hand escaping), and the fact that the supinator longus was atrophied, served to convince me of the peripheral origin of the attack.

CHAPTER III.

LEAD NEURITIS.

I shall next briefly consider the features of lead paralysis, which I am tempted to do by the fact that I have recently had the opportunity, very seldom got, of examining the nerves and cord in a typical case of lead drop-wrist. But for this I would not venture to discuss the subject, as I am sure every one is thoroughly familiar with the classical signs of this variety of paralysis. I am unable to give in detail the history and symptoms of the case on which I made a *post-mortem*, as the patient died of pneumonia and granular kidney a day or two after his admission to hospital, and before his case was recorded in the case book. I have, therefore, selected another example of lead paralysis, which resembles very closely the nervous features of the case referred to, and which will serve to illustrate the distribution and behaviour of the paralysed muscles generally in lead palsy.

D. B., a single man, aged 34, an operative at a white-lead factory, was admitted into the Infirmary, on May 6th, 1886, complaining of double dropped-wrist of one month's duration. He stated that he had suffered from lead-poisoning five times. The first three attacks were of the nature of colic. In the fourth, "he had numbness and weakness of the arms," in addition to colic, and in the fifth, which was the most severe, the arms became paralysed.

History of the present (fifth) attack :—He continued under treatment for the fourth attack for about a fortnight, and then returned to the factory and remained at work for about five or six months. He was engaged, at this time, in the washing department, and had to drain away the water from the large tanks after the lead had gravitated to the bottom. His hands and arms were thus constantly in water impregnated with lead. About five weeks before his admission into the Infirmary he noticed, when at work, that the hands, especially the right, were not so strong as usual, and, in consequence, he had a difficulty in grasping. Next day, the third and fourth fingers of the right hand dropped when an effort was made to extend them. In this condition he continued to work for a few days, but at length the hands became so weak that he was compelled to desist. Accordingly, he ceased work on the Tuesday, and on the following Saturday morning he noticed, when he awoke, that both wrists had dropped, rendering the hands almost useless. The left became paralysed without the preliminary loss of power in the third and fourth fingers that characterised the onset of the paralysis in the case of the right hand. In addition to the paralysis he suffered from a sharp attack of lead colic, which lasted for eight days.

On admission, about five weeks after the onset of the paralysis, the following points were noted:—The patient's skin presented the usual earthy, sallow appearance; the lips were pale, and the nails somewhat purple; the temperature was normal, and the pulse was slow—50; there was well-marked double drop-wrist, and the usual exemption of the supinator longus muscles. The flexors appeared to be weaker than in health, but this was obviously due to the disadvantage under which they laboured, owing to the paralysed condition of their antagonists. Marked atrophy of the extensor muscles, with loss of faradic excitability, was also apparent.

This patient was kept in hospital until the 18th of July, when he was discharged very much improved, though the extensors had not entirely regained their power. The treatment consisted mainly in the use of the interrupted current.

About the time this patient was an inmate of the Infirmary, the man to whose case I have referred, was admitted, complaining of typical double lead paralysis (drop wrist), acute pneumonia, and old-standing kidney disease. His paralysis was of about eight weeks' duration, and, while off work (white-lead works) for this and renal dropsy, he was attacked with pneumonia, about six days before his admission. The blue line on the gums was well marked, and it was quite obvious that the paralysis of the extensors was due to lead-poisoning. He died two days after his admission, and a *post-mortem* examination was made, with the following result:—

Pronounced blue line on the gums. Double pneumonia; hypertrophied left ventricle; granular kidneys. The arms were markedly wasted, especially on the extensor aspect. The common extensors were extremely pale and soft, and presented a great contrast to the adjacent red and healthy-looking supinator longus muscles. Portions of the musculo-spiral and posterior interosseous nerves were removed for microscopical examination; the cervical enlargement of the spinal cord was also examined and preserved, as well as portions of the kidney and paralysed muscles.

Pieces of the kidney were sent to Dr. Walker for chemical examination, who reported that the small portion he examined—about a drachm—contained $\frac{1}{7000}$th of a grain of lead.

It was also ascertained that the muscles, healthy and paralysed, contained lead, about the same quantity being detected in both.

The spinal cord was examined microscopically, but no changes were detected.

The posterior interosseous nerves showed some very pronounced morbid conditions. In some sections there was a great increase in the endoneurium, some of the nerve bundles being made up almost entirely of new fibrous tissue. In others, again, the endoneurium was scarcely altered at all, but the axis cylinders were seen to be much swollen. Here and there groups of new axis cylinders replaced the normal structure, forming the greater portion of the bundle. When viewed longitudinally many of the nerve fibres looked like bundles of connective tissue, though, in places, granular masses of neurilemma could be seen (see fig., page 21.)

The musculo-spiral presented in places similar changes, especially small bundles of new axis cylinders and degenerated fibres, but the great bulk of the nerve was practically unaltered. The simplest explanation of the presence of the degenerated fibres in the main trunk of the musculo-spiral was that they were the continuation upwards of the morbid fibres of the posterior interosseous nerve. No change in the endoneurium was to be seen in the musculo-spiral. Microscopical examination of the paralysed extensors revealed little worth noting. As a rule the cross-striation of the muscle fibres was perfect, but, here and there, even with the high power, all trace of striation was lost, and this homogeneous appearance was observed in several fibres lying side by side. Some of the fibres were much reduced in size, causing a striking inequality in bulk. In a few fibres granular degeneration could be detected, but this was by no means common.

In this case, at least, it will be seen that the lesion was peripheral; but whilst I am inclined to the view that lead palsy is generally the result of a peripheral lesion, it must not be lost sight of that some observers, Oppenheim, for example, have described changes in the cells of the anterior cornua of the spinal cord, and that De Watteville and Erb have recently made the suggestion that a functional lesion of the anterior ganglionic cells, insufficient to produce gross changes, may give rise to degeneration of the motor nerve trunks, and therefore, in reality, the primary disturbance from lead intoxication may fall upon the cord. I confess I am opposed to this latter view, and am disposed to regard the peripheral lesion as the essential factor in the production of the paralysis.

It is a well-known fact that lead palsy may extend beyond the upper extremities, indeed, cases are frequently met with in which the leg muscles are attacked, when double *ankle-drop* generally occurs. It is worthy of attention that the tibialis anticus has been spared in many of these cases, a point of interest when the exemption of the supinator longus, in the case of ordinary drop-wrist, is recollected. In some instances, the paralysis is widely dispersed over the muscles of the limbs, trunk, and face, but these are comparatively rare.

As an example of saturnine palsy in which the law that usually regulates the distribution of the paralysis was not observed, the following may prove instructive :—

Henry C., aged 37, a worker in a white-lead factory, was brought to the Royal Infirmary by the police, on December 16th, 1887, having been picked up in a fit. On the night of the 17th, he had five fits, and twelve on the night of the 18th. On regaining consciousness, he stated that some months previously, a few weeks after commencing to work amongst lead, he suffered from colic and arthralgia. He next met with a slight injury to the right hand and developed a poisoned wound near the base of the thumb, the hand becoming inflamed and swelled, the result, in his opinion, of absorption of lead by the wound. A blue line was now to be seen running up the arm, and the glands in the axilla became swollen. He continued to work for a month longer, when he felt a pain along the radial aspect of the back of the right forearm, which was most severe about the head of the radius at the elbow. Very shortly afterwards, the fingers of this hand dropped, with the exception of the index, which remained straight, but more or less fixed. A few weeks later, the left hand became affected but without previous pain.

When he came under my notice his appearance was strikingly characteristic of lead cachexia. The blue line was well marked on the gums. There was no optic neuritis. On attempting to stretch out the fingers, the middle, ring, and little hung in a flexed condition, but the index finger appeared to be fairly straight; there was imperfect drop-wrist, and marked paralysis of the flexors. The supinator longus was paralysed and atrophied. Abduction and adduction of the fingers were lost. Sensibility was practically normal. Both hands were similarly affected. Thus we had paralysis of the muscles supplied by the posterior interosseous, the median, and ulnar nerves. Little or no response could be elicited by either form of electrical current. The legs were not affected; the knee-jerk was normal.

It may be argued that this case was really an example of *septic* neuritis, and I am not prepared to dogmatize on the point, but the evidence of constitutional lead poisoning was undoubted, which, of course, is highly suggestive.

As in the majority of the cases of paralysis from peripheral lesion, no matter what the cause may be, we meet also in lead paralysis with an alteration in the electrical reaction. Faradic contractility is lost early, and, in many cases, the *reaction of degeneration* is observed, but, in my own experience, it is difficult to demonstrate the typical reaction of Erb, loss to both forms of current being the rule.

CHAPTER IV.

BRACHIAL NEURITIS.

The group of cases I am now about to describe is characterised by paresis or paralysis of one or both upper extremities—brachial monoplegia or paraplegia. In some, injury appeared to be responsible; in some, septic absorption; in others, rheumatism; whilst some, possibly, were erratic forms of alcoholic paralysis; and, in not a few, there was a marked absence of any etiological factor whatever. In the great majority the lesion was chiefly motor, and the paralysis was most pronounced in the region of the distribution of the musculo-spiral and ulnar nerves, and the tendency was, undoubtedly, towards recovery.

In the following case it was difficult to assign any cause for the paralysis which affected almost symmetrically certain muscles in both arms, though the rheumatic theory appeared most tempting:—

J. B., aged 39, a strong, robust looking man, engaged in loading ships on the Quayside, was admitted into the Royal Infirmary on June 17th, 1886, complaining of loss of power in both hands, of three weeks' duration. The family history was good. An enquiry into his previous history elicited little that was material to the case. He had not suffered from syphilis, and was by no means a heavy drinker. His work as a Quayside labourer necessarily exposed him in a trying way to the weather. Six weeks before his admission to hospital he experienced an attack of lumbago, for which he was obliged to rest for upwards of three weeks, when the pain, which was ascribed to rheumatism, subsided, and he was able to renew his work. On rising one morning, nearly three weeks before he was admitted as a patient, he felt numbness in the hypothenar eminence and along the ulnar border of the little finger of the right hand. The numbness rapidly extended, and in a few hours affected the ulnar border of the right ring finger, when he discovered that he was unable to extend these fingers. In a few days, the extensor paralysis becoming gradually more and more pronounced, he detected a similar numbness in the hypothenar eminence of the left hand, and now perfect extension of the little finger of this side was impossible. Next day, decided extensor weakness of the ring finger of the left hand was noticed; thus the two fingers of both hands became impaired in precisely the same way, and later, the middle fingers shared the same fate. During the development of the paralysis no pain was complained of.

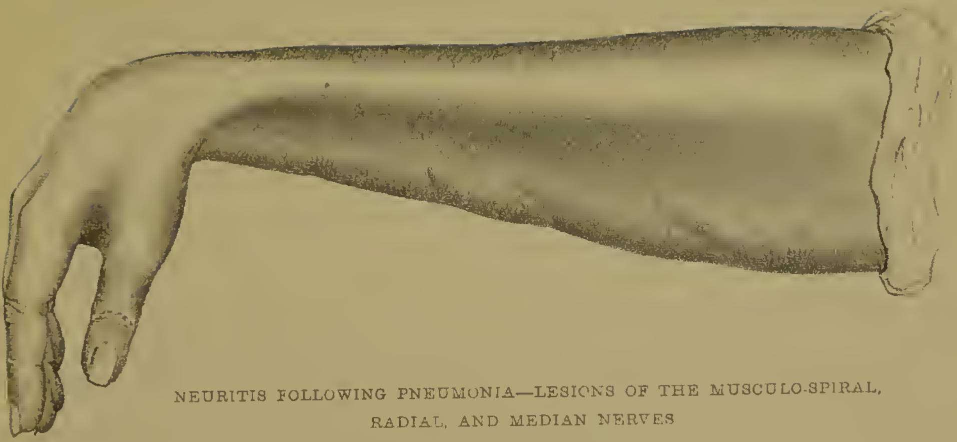
On admission he was seen to be a stoutly-built man of fourteen stones weight, and looked the picture of health. On attempting to stretch out the right hand, the middle, ring, and little fingers remained partially flexed, the first phalanges being extended fully, whilst the second and third were bent so as to give the hand an imperfect claw-like appearance. There was no rigidity, as the fingers could be straightened by passive movements without difficulty. No wasting was observed on the palmar surface, but on the dorsal there were inter-metacarpal depressions, towards the ulnar side especially. The act of separating the fingers was very imperfectly performed. The paralysis appeared to affect the following muscles:—Abductor minimi digiti, opponens minimi digiti, ulnar lumbricales, interossei, adductor pollicis; in other words, accepting Duchenne's explanation of the actions of the smaller muscles of the hand, and Hutchinson's confirmation of the same, the paralysis was limited to the muscles supplied by the ulnar below the articular (wrist) branch. The ulnar flexors of the carpus and little and ring fingers were intact, and the sensory paralysis, which was very carefully tested, was limited to the ulnar portions of the palm and back of carpus, and the ulnar side of the ring finger, together with the little finger. The left hand was affected in almost precisely the same manner, and the anæsthesia was the same. An attempt to extend the fingers resulted in drawing back of the first, and flexion of the second and third meta-carpal bones of the three inner fingers, whilst separation of the fingers was interfered with, and it was with difficulty he could approximate the thumb and little finger. The paralysed muscles responded very feebly when tested with the interrupted current; the continuous current was out of order during the short time the patient remained in hospital. Very rapid improvement took place, and, though the course of treatment (faradism and massage) was very brief, the paralysed muscles soon regained their functional activity.

In the following case, gout, or perhaps more correctly speaking, rheumatism, was probably the cause of the attack of neuritis:—

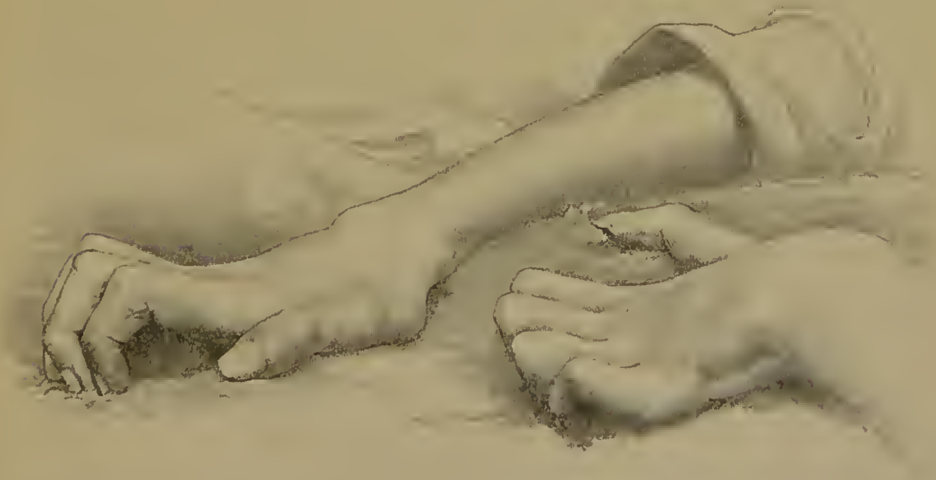
Eliza M., aged 64, was made an in-patient of the Infirmary on March 24th, 1886. For upwards of two years before the paralytic symptoms appeared, she suffered, from time to time, from painful swelling and redness of various joints—sometimes the ankles, sometimes the phalangeal joints were affected—the inflammation only lasting a day or two. Four months before she came under observation, she discovered, on waking one morning, that the middle finger of the right hand was "contracted," red, hot, and swollen. This was poulticed, and the doctor incised it on the dorsal surface, but no pus escaped. It was afterwards painted with iodine, but soon the same difficulty in extending the other fingers of the same hand was experienced, and then the finger joints of the left hand grew painful, and their flexors became weak. On admission, the fingers of the right hand were contracted and rigid, and resisted passive movements, especially the middle and ring fingers. The common extensor was atrophied, and the flexors of the fingers were considerably weakened, but flexion and extension of the carpus were, practically, unaffected. The extensors of the left hand were similarly affected, though to a slighter degree. The dorsal surfaces of both fore-arms were anæsthetic. The paralysed muscles showed a decided quantitive diminution when tested electrically. Iodide of potassium was ordered, and massage and faradism employed, with decided benefit.

In the case of F. S., aged 56, a male attendant in a lunatic asylum, who was placed under my care by my friend Dr. T. W.

McDowall, the peripheral monoplegia occurred during convalescence from right-sided croupous pneumonia. Eighteen months before this illness he contracted syphilis. About the tenth day of the pulmonary attack, he was seized with severe burning pain in the right arm, about the insertion of the deltoid, whence it passed down the inside of the forearm to the palm of the hand. In a few hours the pain subsided, leaving the extensors of the wrist and fingers paralysed; whilst sensation was lost on the back of the hand, the little finger and adjacent side of the ring finger alone escaping. It was now observed that the muscles supplied by the musculo-spiral, median, and radial nerves were paralysed. By degrees some improvement took place, and when he was admitted into the Infirmary, three months after the onset, the following points were recorded:—The general health was good. The right forearm was paralysed and wasted. The wrist was flexed, and the hand hung as in lead palsy. He could extend the carpus slightly, but the extensors of the fingers were almost completely paralysed, the index finger being most affected. The muscles supplied by the ulnar nerve seemed normal. He had no difficulty, for example, in separating the fingers of the paralysed hand. There was marked paralysis of the flexors of the fingers, more especially on the radial side. The supinator longus was paralysed, and the deltoid, biceps, and triceps were feeble. All the paralysed muscles were more or less atrophied, though the wasting chiefly affected the flexors in the neighbourhood of the radius, the supinator longus, and the extensors of the fingers. The accompanying drawing, (see fig.), kindly made for me by Dr. Baigent, one of our House Surgeons, shows well the marked wasting of the flexors on the front of the forearm, as well as the position usually assumed by the hand. The areas of skin supplied by the median and radial nerves were decidedly anæsthetic, but those supplied by the cutaneous branches of the musculo-spiral were normal. The paralysed muscles all showed a marked lowering of the galvanic and faradic irritability, and, in the case of the most paralysed, the strongest currents elicited no response. Treatment—massage and faradism—improved considerably the bulk



NEURITIS FOLLOWING PNEUMONIA—LESIONS OF THE MUSCULO-SPIRAL, RADIAL, AND MEDIAN NERVES



FROM A CASE OF GENERAL MULTIPLE NEURITIS FOLLOWING SCARLET FEVER—BOY AGED 8.

and power of the affected muscles, with the exception of the flexor of the index finger, which resisted weeks of diligent and energetic manipulation. He left the Infirmary, on the whole, considerably improved, but with what was feared might turn out to be a permanently damaged hand, owing to the condition of the index finger, though I have since learned that, after the lapse of time, further improvement has taken place.

The suddenness of the onset of the attack in this case might, no doubt, suggest at first sight, a cerebral lesion, and indeed, in some cases of peripheral palsy, the diagnosis is by no means easy. The following case I quote with the object of emphysizing this difficulty :—

Mrs. A., aged 57, consulted me recently for weakness of the muscles of the left forearm. She stated that her general health had not been good for some months past, and that she had come to Newcastle for change of air, and had a long railway journey two days previously, when she suffered very much from the cold, the weather being very trying at the time. On rising to dress, on the morning following her arrival, she felt giddy, and observed a numb feeling in the left forearm and hand. A few minutes after her attention had been called to the limb, she discovered, on attempting to use it, that it was almost powerless. Being anxious not to alarm her friends, she made light of the attack and ascribed it to cold, but, as the day wore on, it became apparent that the numbness, with a certain degree of "stiffness," had extended to the left leg, interfering with locomotion. I saw her the day after the paralysis appeared. She was a feeble looking woman, and was evidently apprehensive of approaching danger. The left hand was almost completely paralysed, especially the little and ring fingers which remained in a semiflexed position. The index and middle fingers could be flexed and extended slightly, but with markedly diminished power. The lumbricales and interossei supplied by the ulnar were paralysed. The supinator longus was weak, and the remaining muscles seemed to be more feeble than in health. Sensibility in the median and ulnar regions of the hand was impaired, especially as regards painful impressions.

There was some pain complained of along the back of the forearm. There was no appreciable weakness of the leg. *On both sides the knee-jerk was exaggerated.* The pulse was hard, and the second sound of the heart was high pitched and accentuated. There was a fair quantity of albumen in the urine. There was no facial paralysis, and the tongue, which was clean, was protruded straight. The subsequent events, as the case progressed, proved the peripheral origin of the paralysis, a diagnosis that was founded in the first instance upon the partial and special distribution of the paralysis, the pain and anæsthesia that accompanied the motor weakness, the total absence of facial, tongue, or speech impairment, and the fact that the paralysis did not extend beyond the area first affected; whilst the later events decidedly established the diagnosis.

This case must, I think, be regarded as a striking example of peripheral palsy arising from exposure to cold, and may, therefore, be very properly called rheumatic. The paralysis in the case I am about to adduce depended, I believe, upon septic absorption, and, as I am inclined to think that septicæmia, in some shape or form, is a commoner cause of peripheral nerve lesions than is generally supposed, I wish to bring the matter particularly under the notice of surgeons who, necessarily, have the opportunity of watching the development of the mischief.

A young woman, aged 18, of robust constitution, was sent to me by Mr. Rutherford Morison, whose acquaintance with the case was subsequent to the full development of the lesion, with paralysis and contraction of the fingers of both hands. I first saw her on the 9th June, 1885, and she stated that her disabled condition had its origin in a cut she received on the end of the middle finger of the left hand in January, 1884. The wound suppurated, and was treated by a chemist, who applied caustic. In a little while, she noticed that the finger was becoming paralysed and contracted. Then the ring finger became weak, flexed, and rigid, and later, the index and little fingers shared the same fate. Some months after the onset of the paralysis the extensors of the fingers of the right hand failed, and soon the fingers were firmly flexed in the palm, as in the case of the right hand. In December, 1884, the surgeon in charge of the case amputated the injured finger at the metacarpo-phalangeal articulation, and very shortly afterwards the thumbs of both hands became firmly flexed.

When she came under my notice, her condition was as follows:—The fingers on both sides firmly clenched, and the extensors completely paralysed, and markedly wasted, whilst their faradic contractility was entirely abolished. The flexors were also much affected, though not quite powerless, and they reacted fairly to the interrupted current. The paralysis and contracture were most marked on the left side. There was no anæsthesia, but the patient complained constantly of a pain along the course of the left radial nerve.

After repeated efforts to straighten the flexed fingers, under chloroform, and the diligent employment of faradism and massage for many months, we had the satisfaction of witnessing great improvement in the disabled limbs. First, the electrical contractility of the paralysed extensors returned, and then slight power was regained over these muscles, and, by the time she left the Infirmary, the left hand was a fairly useful member, whilst the right was considerably improved, though by no means restored.

It is interesting to note the progress of the lesion—first, the posterior interosseous nerve on the right side, then the corresponding nerve in the left forearm. This, I may remark, is a march of events very often observed in peripheral palsies, and the question arises, does the lesion travel across the cord and affect the corresponding fibres in the opposite cornu, or are the conditions in no sense continuous?

It may be instructive to record the following case in which the peripheral paralysis arose from the hypodermic injection of ether into the left arm:—A man, aged 60, was admitted to the Royal Infirmary, for compound comminuted fracture of the leg, necessitating amputation of the lower third of the thigh. During the operation, he showed signs of collapse, and ether was administered, the needle of the syringe being introduced deep into the outer part of the left arm. Six days after the operation, left drop wrist occurred, and all the features of severe paralysis of the muscles supplied by the musculo-spiral developed. He recovered well from the operation, and, after upwards of two months treatment by massage and faradism, he had practically regained the use of the paralysed and atrophied muscles.

CHAPTER V.

PROGNOSIS AND TREATMENT.

It is of considerable importance, in attempting to predict the result of an attack of paralysis from peripheral neuritis, to determine the cause of the mischief, as the etiology and prognosis hang close together. The most serious cases met with in this country, as far as our present information carries us, are those arising from alcohol. Of these many die, an untoward result that is to be ascribed, as a rule, to the persistent use of the stimulant, even after the patient has come under treatment. Nor is the profession entirely free from blame in this matter, as alcohol is often ordered with the view of "keeping up the strength," when probably it is the one agent to be feared. It must not be supposed that I regard the withdrawal of the stimulant as an easy task, for no doubt the difficulties are great, as the female toper generally finds a means of indulging her tastes; and not uncommonly a servant, or some friend who has been in the secret, and may perhaps have aided in the consumption, will contrive to defeat the doctor's orders.

The prognosis in alcoholic cases embraces the question, will the patient live? as well as the prospect of his regaining the use of the affected limbs. I am disposed to think that a favourable view may be taken on both questions in almost every case, provided the stimulant be stopped, and suitable treatment adopted. It is true that, in some cases, the disease will advance in defiance of all our efforts, but here the danger has usually escaped recognition until it is too late. In the case of a man, aged 35, whom I recently saw with Dr. Todd, of Gateshead, the paralysis and cerebral symptoms ran an acute course, the patient dying three weeks from the onset of the paraplegia, notwithstanding the stimulant was at once discontinued. This, I believe, to be the most rapid case on record. The fatal cases that have occurred in my practice now amount to eleven, three men and eight women. Of these, one was determined by pneumonia, one by cirrhosis of the liver, whilst the remaining nine could only be ascribed to the unchecked progress of the disease. The risk to life would appear to be in direct ratio to the gravity of the cerebral symptoms, given loss of memory, a fatuous and perhaps an emotional manner, and other decided evidences of brain disturbance, along with paralysis of the legs and arms in an inveterate toper, and the prognosis is exceedingly grave, especially if the alcoholic stimulant be persevered in.

When considering the patient's chance of regaining the use of the paralysed members, it must not be lost sight of that recovery is often very protracted and tedious, and that many months may elapse before definite improvement can be recognized. And further, after the case has commenced to progress favourably, even occasional indulgence in ardent spirits may induce a decided relapse, and materially interfere with recovery. On more than one occasion, on observing that the patient was not so well (the pains or the paralysis more pronounced) I have suggested to the nurse in charge, as an explanation, that the forbidden stimulant was being conveyed in clandestinely, and, on enquiry, the surmise has proved to be correct.

A case that came under my notice some years since was peculiarly interesting in this connection; it was just at the time when the true pathology of this form of paralysis was beginning to dawn upon us. An elderly woman was admitted repeatedly into the Infirmary with alcoholic neuritis, and she commonly remained about six or eight weeks at a time in the Institution, returning home, as a rule, considerably improved, but only to be readmitted in a few weeks in a worse condition. In this way nearly eighteen months passed, during which she was some four or five times in Hospital. Her friends then procured her admission into the Incurable Hospital, with the result that, at the end of twelve months, she was completely cured. The truth was, that between the periods of her residence in the Infirmary, she had freely indulged her appetite for liquor.

In all cases of peripheral neuritis the fact should be recognized that the element of time is of the greatest importance; many condemned cases have been known to recover after the lapse of months, when both doctor and patient have given up all hope. This is especially true of the alcoholic cases, though probably it is equally so of all the chronic varieties of peripheral lesions. In my experience, the cases that prove most obstinate and seem to justify the most serious prognosis, so far as the local condition is concerned, are severe saturnine palsy; so-called gouty cases in which the joints have become stiff; some septicæmic cases, and cases arising from injury, which are difficult to separate from the same category; and, of course, severe traumatic lesions. On the other hand, the rheumatic, the syphilitic, the diphtheritic, and a great group of cases characterised by paralysis of groups of muscles of one or both upper extremities, concerning whose etiology we have little or no information, usually do dwell. Nor do I think, one would be justified in excluding the majority of cases of general multiple neuritis from this series, though I am bound to say some of my cases turned out most disappointing, a few muscles here and there remaining paralysed and atrophied.

I have stated that the prognosis is usually good in diphtheritic paralysis, and, no doubt, most cases recover, but it is important to recollect that a certain proportion die from paralysis of the laryngeal muscles, or of the diaphragm or other respiratory muscles, or from cardiac failure, and that when everything points to a favourable termination. Three cases that came under my notice recently, succumbed in the most unexpected way, so that I am disposed, until a certain stage is passed, to speak very guardedly of the prospects of these cases.

Although I have laid great stress upon the element of time as an important factor in the recovery of paralysis from peripheral neuritis, I wish to emphasize, with equal force, the value of treatment. Except, perhaps, the almost miraculous effect that attends the treatment of some functional disorders by massage isolation, etc., there is nothing that furnishes more satisfactory results than active and assiduous attention to detail in the treatment of peripheral lesions. The prognosis is, indeed, largely influenced by treatment, which is more than can be said for the majority of nerve affections. The indications for treatment may be stated to be (*a*) the general management of the affection, (*b*) the relief of pain, (*c*) the stimulation and development of paralysed and atrophied muscles, (*d*) relieving fixed joints and contracted tendons. By the general management of the disease, I mean the treatment of the condition upon which the neuritis depends, as well as the efforts directed towards the abatement of the inflammation of the nerve trunks. For the latter purpose I have great faith, when not otherwise contra-indicated, in the use of mercury, *locally* and *internally*. The blue ointment may be gently rubbed along the course of the nerves, or the oleate with lanolin applied. When the pain is severe, morphia or cocaine, injected hypodermically, are usually required, or else opium in some form by the mouth, for, unfortunately, less potent anodynes will seldom give relief, though belladonna applications and hot fomentations are sometimes of service. As soon as they can be borne, massage and electrical stimulation ought to be resorted to, and the form of current selected to which the muscles respond best. I would once more put in a claim for patient perseverance in conducting this plan of treatment, for what appears to be the most hopeless case, will often show surprising results

The joints show a peculiar tendency in many cases of neuritis to become stiff; especially is this noticeable in cases arising from injury, septic conditions, gout, and rheumatism. Under these circumstances, it may be necessary to break down adhesion, and for this purpose chloroform will usually be required.

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